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The effect of thoracentesis on lung function and transthoracic electrical bioimpedance

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This study aimed to determine the relationship between improvement in lung function and changes in transthoracic electrical bioimpedance (TEB) after thoracentesis in patients with pleural effusions.

Fifteen patients with pleural effusions due to either malignant (n=8) or cardiac (n=7) diseases were included. Pulmonary function was assessed before and after thoracentesis. During thoracentesis the patients were monitored with TEB.

Using linear correlation analysis, the increases for each litre of aspirated thoracic fluid were: forced expiratory volume in 1 s (FEV_1) 0·26 l; forced vital capacity (FVC) 0·33 l; total lung capacity (TLC) 0·58; and the lung diffusing capacity (DLCO); 2·4 ml min^{-1} mmHg^{-1}. Baseline impedance increased by 2·3 Ohm l^{-1} aspirated thoracic fluid. The relative increase in baseline impedance was twice as high for patients with cancer as for patients with heart failure (P<0·05). We found only minor changes in systolic blood pressure and mean arterial pressure.

The improvements in diffusing capacity, airflow, and lung volumes after thoracentesis are correlated to an increase in baseline impedance, but changes are dependent on the primary disease.

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The basis for the clinical observation of relief of dyspnoea experienced by patients after thoracentesis has been disputed. Thoracentesis has been associated with increasing lung volume (1) and increases in arterial oxygen tension (2), but the reported changes in lung volume have been inconsistent and small. The changes have been characterized as being unable to explain the clinically well-known relief of dyspnoea in these patients (3), and even lead to the assumption that it could be a placebo effect (1). Other studies have attributed the relief of dyspnoea to changes in mechanical variables of respiratory muscle function (4). Changes in thoracic fluid volume and cardiac output can be assessed by transthoracic electrical bioimpedance (TEB) (5). The method has found clinical use in monitoring intensive-care patients, especially those with hydrothorax and pulmonary oedema. It cannot, however, distinguish between changes in intravascular and extravascular fluid volumes (6). The aim of this study was to compare changes in pulmonary function with changes in TEB related to thoracentesis. Pulmonary function was assessed before and after thoracentesis. The changes were compared with changes in haemodynamic variables and the baseline impedance, which reflects the amount of fluid present in the thorax.

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Materials and Methods

PATIENTS

Sixteen patients, 11 men and five women [median age 67 years (range 48–77 years)] with pleural effusions due to either cardiac (one woman and six men) or malignant diseases (four women and five men) participated after informed consent. Two patients did not complete both measurements of lung diffusing capacity due to fatigue. One male patient with lung cancer was excluded from the study as he developed pneumothorax during thoracentesis. Four of the pleural effusions were sanguinary, the remaining 12 serous. All pleural effusions were unilateral. All patients were in a clinically stable state at the time of examination. The local ethics committee for Copenhagen approved the study.

METHODS

To use an electrical metaphor, the thorax can be viewed as a transducer shaped like a truncated cone, with a

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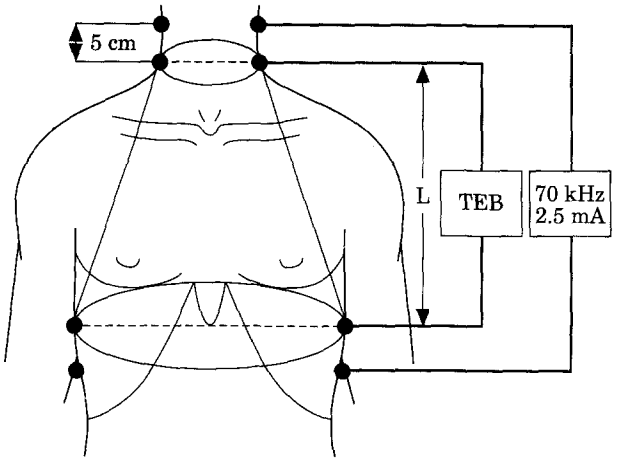

FIG. 1. Schematic representation of the thorax with transmitting (2·5 mA and 70 kHz) and receiving (TEB) electrodes. L is the length of the thorax and the distance between the inner electrodes. The truncated cone represents the theoretical volume of the electrically participating tissue.

steady-state baseline impedance (Z_0 in Ohm) (7). This baseline impedance depends on an intrathoracic volume conductor, i.e. fluid or blood. The physical volume of the electrically participating tissue is based on the size of the patient and can be expressed as a constant (C) dependent on the length of the patient's thorax (L) determined from a normogram of height and body weight. $C=L^3/4{\cdot}25$ (Fig. 1) (8).

The impedance changes during the cardiac cycle because of the pulsate variations in thoracic aortic blood flow. The maximum rate of impedance change $(dZ/dt)_{max}$ (Ohm s^{-1}) within one cardiac cycle is proportional to the peak ascending aortic blood flow during systolic upstroke (Fig. 2). This change in impedance can be related mathematically to stroke volume (SV) by the equation (7):

$$SV=C*VET*(dZ/dt)_{max}/Z_0, \quad \text{(equation 1)}$$

where VET is the ventricular ejection time (Fig. 2).

TEB was measured with a BoMed (BoMed Medical Manufacturing Ltd, Irvine, CA, U.S.A.) impedance cardiograph NCCOM-3 (8). Impedance was measured across the thorax between four electrodes placed around the basis neck at the level of the lung apex and four electrodes placed around the chest at the level of the xiphoid process corresponding to the level of the lung basis (Fig. 1). A high frequency alternating current of 2·5 mA at 70 kHz was sent between the two outer sets of electrodes and the impedance signal was measured between the inner sets of electrodes. A built-in microprocessor provided calculations of VET, heart rate (HR), stroke volume (SV), and cardiac output (CO) as a mean value of three readings each consisting of 12 cardiac cycles with acceptable impedance readings (8).

Thoracentesis was performed with the patient in a fixed sitting position. The pleural fluid was aspirated with a needle, inserted under local anaesthesia through the eighth or ninth intercostal space. Aspiration of thoracic fluid was continued until the patients developed symptoms from

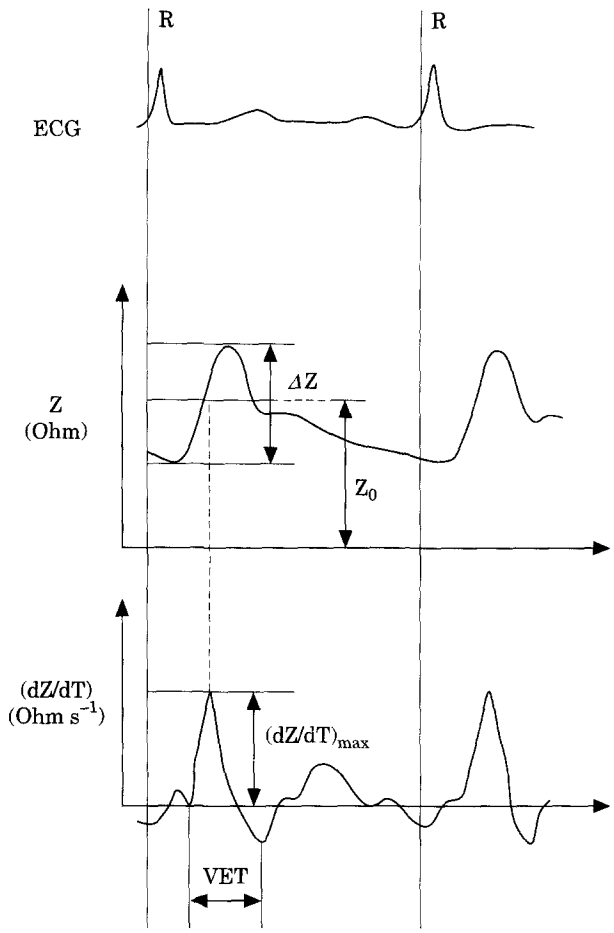

FIG. 2. Relationship between cardiac cycle as represented by ECG, transthoracic impedance (Z), and the first differential of Z (dZ/dt). The R wave on the ECG is used to trigger the impedance readings. VET is the ventricular ejection time.

pleural irritation such as chest pain or coughing or until no more fluid could be withdrawn. Thorax radiographs taken shortly after thoracentesis showed no signs of pulmonary oedema or pneumothorax in any of the patients except the one excluded because of pneumothorax.

TEB recordings were performed for each 500 ml of aspirated pleural exudates as well as before and at 5 and 10 min after thoracentesis. Stable values of impedance readings were obtainable for each step after 1 min.

Blood pressure was measured along with TEB with an arm cuff and a sphygmomanometer. Spirometry and lung diffusing capacity assessment were performed using a Sensor Medics 2450® Computerised Pulmonary Function Laboratory (CA, U.S.A.). Pulmonary function testing of forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), total lung capacity (TLC), and lung diffusing capacity (D_LCO) was performed immediately prior to and between 1 and 2 h after thoracentesis according to the recommendations of the British Thoracic Society (9). The highest of two test results that differed by no more than 3% was used as the final test result. TLC and D_LCO were determined with the single breath technique after inhalation

TABLE 1. Changes in TEB variables and blood pressure before, after, and during thoracentesis

	pre	δ 500 ml	pre	δ 1000 ml	pre	δ 1500 ml	pre	δ 2000 ml	δ 10 min
n	15		12		8		4		15
Z_0 (Ohm)	24·4	25·4***	22·4	24·6***	19·9	22·5**	13·9	17·2(*)	26·0****
HR (min^{-1})	93	84*	92·5	87·5	89	84·5	84	88·5	91(*)
dZ/dt_{max} (Ohm s^{-1} 10^{-3})	0·82	0·84	0·77	0·83	0·67	0·82(*)	0·51	0·63	0·93(*)
SV (ml)	51	54	49	51	49	49·5	39	54	51
CO ($l\ min^{-1}$)	5·5	4·6	4·8	4·7	4·5	4·5	4·3	4·0	4·8
BPS (mmHg)	150	140	158	140*	158	143(*)	140	132	140***
BPD (mmHg)	90	83*	88	80*	90	83(*)	80	80	80
MAP (mmHg)	108	103*	109	102***	114	103*	100	98	105***

Median values are given for each 500 ml of aspirated fluid and the values before thoracentesis (pre). Statistical method: Wilcoxon matched-pairs test.
n: number of patients; Z_0: baseline impedance; HR: heart rate; SV: stroke volume (see equation 1 in the text); CO: cardiac output=SV*HR; BPS: systolic blood pressure; BPD: diastolic blood pressure; MAP: mean arterial blood pressure=(1/3*(BPS − BPD)+BPD).
(*)=$P<0·1$; *=$P<0·05$; **=$P<0·02$; ***=$P<0·01$; and ****=$P<0·001$.

TABLE 2. Median values and ranges for lung function variables before and after thoracentesis

	Before thoracentesis	After thoracentesis	Median change	Median change (%)
FEV_1 ($l\ s^{-1}$)	1·10 (0·52–2·55)	1·40 (0·95–2·65)	0·21 (−0·07–0·77)**	17·3 (−6·1–148·1)
FVC (l)	1·86 (1·03–4·00)	2·05 (1·50–4·24)	0·42 (0·02–0·98)**	18·3 (0·6–73·2)
VC (l)	2·18 (1·15–4·29)	2·12 (1·47–4·50)	0·28 (−0·28–0·82)**	14·2 (−11·7–60·9)
RV[a] (l)	2·38 (1·17–4·32)	2·56 (1·40–3·73)	0·13 (−0·59–1·22)*	5·5 (−13·7–104·3)
TLC[a] (l)	4·85 (2·56–7·84)	5·11 (2·67–7·75)	0·26 (−0·09–1·49)**	5·8 (−1·15–49·2)
*D*LCO ($ml\ min^{-1}\ mmHg^{-1}$)	12·1 (7·3–26·6)	14·1 (10·3–29·9)	1·09 (−1·60–6·81)*	9·0 (−8·0–93·3)
Volume of aspirated pleural fluid (l)				1·25 (0·13–2·30)

Statistical method: Wilcoxon matched-pairs test.
**Difference is significant at the 0·01 level (2-tailed). *Difference is significant at the 0·05 level (2-tailed).
[a]*n*=13.

of a gas mixture containing carbon monoxide and helium with the latter as an indicator of the dilution. All patients were free from chest pain at the time of the second pulmonary function test.

DATA ANALYSIS

Linear regression analyses between the volume of aspirated pleural effusion, baseline impedance, and lung function variables were performed.

Changes in TEB, mean arterial pressure, blood pressure, and lung function variables before and after thoracentesis were compared using the Wilcoxon non-parametric test for paired data. The comparison of changes in Z_0 between groups of patients was made using the Mann–Whitney *U*-test.

Results

The median amount of aspirated thoracic fluid was 1250 ml (range 325–2300 ml). All patients experienced a relief of dyspnoea after thoracentesis. Both relief of dyspnoea and changes in lung function variables after thoracentesis were independent of underlying ventilatory defects.

Table 1 shows changes in central haemodynamic variables and Z_0 for each 500 ml amount of aspirated thoracic fluid as well as values obtained 10 min after thoracentesis. Baseline impedance increased significantly with each 500 ml of aspirated thoracic fluid. There were no changes in HR apart from a temporary decrease after aspiration of the first 500 ml of thoracic fluid. The patients had slight but significantly lower systolic and mean arterial blood pressure 10 min after thoracentesis, while diastolic blood pressure showed a trend towards a decrease.

Table 2 shows the results of the lung function test before and after thoracentesis. The patients had a significant increase of 5·5–18% from baseline values in the reported lung function variables.

Figure 3 illustrates the physiological effect on or therapeutic gain of lung function variables and changes in Z_0 when aspiring pleural effusions. From a visual evaluation of Fig. 3, it appears that there is a larger relative beneficial

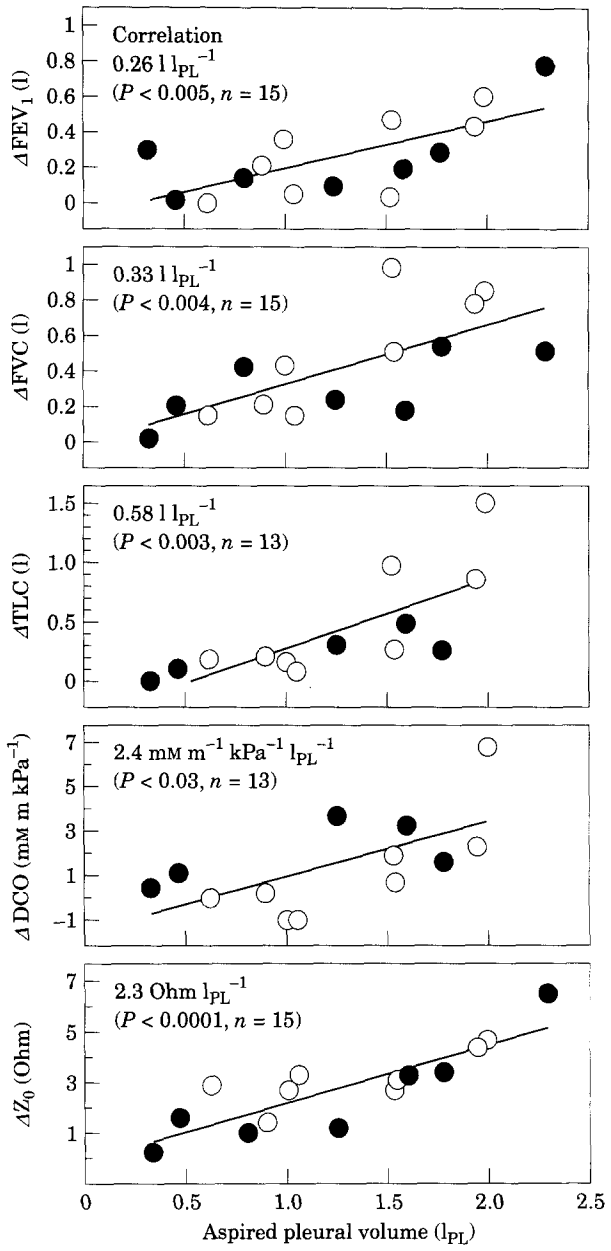

FIG. 3. Effect of aspirated pleural volume on changes in baseline impedance (Z_0), forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), total lung capacity (TLC), and lung diffusing capacity (D_LCO). Slopes and significance levels are derived from a linear regression model. ●: Heart patients, ○: cancer patients.

effect on TLC and D_LCO in patients with pleural effusions over 1 l compared to patients with smaller pleural effusions. FEV_1, FVC, and Z_0, however, seem to be dependent on the aspirated volume of pleural fluid in a more linear fashion. Correlation coefficients between the aspirated volume of thoracic fluid, changes in lung function variables and changes in Z_0 are shown in Table 3.

The transfer coefficient (D_LVa) did not increase as five out of eight of the patients with malignant diseases had declining values of D_LVa after thoracentesis (three of these five patients had primary lung cancer).

There was no correlation between the haemodynamic variables and the aspirated amount of thoracic fluid volume. Even though $(dZ/dt)_{max}$ showed a tendency towards an increase after thoracentesis, no correlation was found between $(dZ/dt)_{max}$ and Z_0 or the amount of aspirated thoracic fluid.

Equal amounts of thoracic fluid were aspirated in patients with cancer [median 1·29 l (range 620–2000 ml)] and heart disease [median 1·25 l (325–2300 ml)]. However, cancer patients had almost twice the increase in baseline impedance [median 2·8 Ohm (range 2·4–4·5 Ohm)] as patients with heart disease [median 1·3 Ohm (range 0·1–6·7 Ohm)]. This was seen most consistently for the percentage changes in Z_0. Cancer patients had a 16% increase in Z_0 per litre of aspirated fluid in contrast to 7% in heart patients ($P<0·05$). There were no significant differences in changes in Z_0 when patients were divided into groups according to gender, body weight, height, obese versus lean, or type of respiratory disease (restrictive/obstructive).

There was no change in baseline impedance between measurements performed at the end of thoracentesis and those performed 10 min after the procedure.

Discussion

Thoracentesis was closely correlated to improvements in lung function and an increase in baseline impedance. In general, studies of the physiological effect of aspirating pleural effusions have been small with inconsistent results. It has even been suggested that the relief of dyspnoea could be a placebo effect (1). This led Estenne *et al.* to examine the mechanical variables of lung function (4). They concluded that the relief of dyspnoea following thoracentesis primarily is due to a reduction in the volume of the thoracic cage, allowing the inspiratory muscles to operate at a more advantageous part of their length–tension curve. On the other hand Mahler *et al.* (10) have found the relief of dyspnoea to be well correlated with changes in spirometric values.

The present data clearly demonstrates that thoracentesis is related to an increase in respiratory flow, volume and diffusing capacity. Our data provide a basis for estimations of improvement in lung function variables from the amount of aspirated thoracic fluid. The most marked improvement in lung function was found in FEV_1 and D_LCO. These findings support both the conclusion of Estenne *et al.*, as mentioned above and Perpina *et al.* (2)), who found major improvements in gas exchange after thoracentesis. However, thoracentesis appears to be of greatest benefit to D_LCO and TLC when the aspirated fluid volume exceeds 1 l. Light *et al.* have found a similar threshold value for VC at 800 ml of removed fluid, regarding the correlation between changes in VC and pleural pressure (11). These findings indicate that the relief of dyspnoea when aspirating small fluid volumes (i.e. less than 1 l) is mainly due to a reduction in the volume of the thoracic cage, facilitating an

TABLE 3. Pearson correlation matrix between aspirated volume of pleural fluid (volume) and changes in lung function variables and baseline impedance (Z_0)

	Volume	$dFEV_1$	dFVC	dVC	dRV^a	$dTLC^a$	dD_LCO^a
$dFEV_1$	0·68**						
dFVC	0·70**	—					
dVC	—	—	0·51*				
dRV_a	—	—	—	—			
$dTLC^a$	0·75**	—	0·84**	0·66*	—		
dD_LCO^a	0·61*	—	0·47(*)	0·52(*)	—	0·78**	
dZ_0	0·85**	0·68**	0·50(*)	—	—	0·68*	

**, Correlation is significant at the 0·01 level (2-tailed); *, correlation is significant at the 0·05 level (2-tailed); (*), trend towards correlation at $0·05 < P < 0·10$; −, no correlation.
[a]$n=13$.

increase in FVC and FEV_1 due to improved function of the respiratory musculature. Only larger volumes of aspirated fluid result in increases in TLC.

Other authors have described a linear correlation between changes in Z_0 and changes in pleural effusion or central blood volume in both humans (12) and in dogs (13). An even closer correlation between changes in Z_0 and changes in lung function variables may have been demonstrated had the impedance readings been extended to the time of lung function measurement (14). Although there is a significant correlation between changes in baseline impedance in Ohms and changes in the amount of thoracic fluid, it is not possible to predict the exact amount of thoracic fluid from baseline impedance readings (15). However, Saunders *et al.* (6) have found transthoracic electrical bioimpedance assessment useful as a non-invasive method of monitoring changes in the amount of thoracic fluid in intensive-care patients. Also, Campbell *et al.* (16) have reported similar findings using a tomographic technique based upon changes in thoracic impedance. Our data support these findings and provide a direct estimate of the amount of fluid accumulated with a given change in baseline impedance. However, the primary disease has a significant impact on the relative changes in Z_0 as a function of the aspirated volume of thoracic fluid. This can be interpreted as a result of patients with heart failure having a higher overall fluid content in the thorax than patients with malignant diseases, because of an increased filling of the vascular bed of the lungs due to a backward failure (17). Thus, the smaller relative changes in Z_0 per amount of aspirated fluid volume in patients with heart failure could be explained by the lesser relative contribution of the aspirated fluid volume to the total impedance.

As baseline impedance (Z_0) had changed during thoracentesis, an unchanged SV under these conditions in fact reflects an increase in $(dZ/dt)_{max}$ (see equation 1). In spite of a tendency towards an increase in $(dZ/dt)_{max}$ after thoracentesis, no correlation was found between $(dZ/dt)_{max}$ and Z_0 or the aspirated amount of thoracic fluid. Thus, our data are not conclusive regarding statements of changes in cardiac output as a function of the aspirated thoracic fluid volume. The decrease in systolic and mean arterial pressures seen 10 min after thoracentesis may be due to relief of distress related to the procedure, rather than its effect. The decline in mean arterial and systolic blood pressure and the trend towards a lower heart rate after aspiration of the first 500 ml of fluid may be seen as a parallel to the vagal response when perforating the peritoneum and/or relief from thoracic fluid compressing the heart.

In conclusion, we have found a close correlation between the drying effect of thoracentesis and changes in both baseline impedance of the thorax and subsequent improvements in pulmonary airflow, lung volume and lung diffusing capacity. The present study provides a basis for estimating the magnitude of the physiological effect of a given amount of aspirated fluid volume. However, the changes in Z_0 appear to be dependent on the disease of the patient, reflecting the overall fluid content of the thorax.

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